Journal of Affective Disorders 134 (2011) 280–287

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research report

Community attitudes to genetic susceptibility-based mental health interventions for healthy people in a large national sample

Alex Wilde [a,c,*], Bettina Meiser [b], Philip B. Mitchell [a,c,f], Peter R. Schofield [d,e,f]

[a] School of Psychiatry, University of New South Wales, Sydney, Australia
[b] Prince of Wales Clinical School, University of New South Wales, Sydney, Australia
[c] Black Dog Institute, Sydney, Australia
[d] Neuroscience Research Australia, Sydney, Australia
[e] School of Medical Sciences, University of New South Wales, Sydney, Australia
[f] Brain Sciences UNSW, Sydney, Australia

ARTICLE INFO

Article history:
Received 25 March 2011
Received in revised form 20 June 2011
Accepted 20 June 2011
Available online 16 July 2011

Keywords:
Major depressive disorder
Genetic risk information
Health behaviours
Primary prevention

ABSTRACT

Background: Despite an apparent high interest in predictive genetic testing for common multifactorial disorders, few data describe anticipated health behaviour as a consequence of such testing.
Methods: A large population-based public survey with community dwelling adults (N = 1046) ascertained through random digit dialling. Attitudes were assessed via structured interviews.
Results: Intention to start therapies or courses to learn to develop better strategies to cope with stress (80%) was significantly and positively associated with self-estimation of risk for major depressive disorder as higher than average (ß = 0.12, p = 0.001); endorsement of family environment as a causal attribution (ß = 0.11, p < 0.001); and endorsement of gene–environment interaction as a causal mechanism of mental illness (ß = 0.12, p = 0.017). Intention to modify potential life stressors (84%) was significantly and positively associated with self-estimation of risk for depression as higher than average (ß = 0.07, p = 0.029); endorsement of 'abuse' as a causal attribution (ß = 0.10, p = 0.003); and endorsement of 'gene–environment interaction' as a causal mechanism (ß = 0.10, p = 0.002).
Limitations: The hypothetical nature of the genetic risk scenario may have weakened participants' sensitivity to the potential personal impact of such a genetic test result.
Conclusions: Perceptions that modifiable environmental factors strongly contribute to overall risk of major depressive disorder appeared to drive willingness to engage in risk-modifying interventions in the hypothetical scenario of a genetic predisposition. Our results suggest that screening for genetic risk in consort with environmental risk factor assessment has potential community acceptability and clinical value as an early intervention and preventive tool for high risk groups.

© 2011 Elsevier B.V. All rights reserved.

1. Introduction

Despite an apparent high interest in predictive genetic testing for susceptibility to common multifactorial disorders amongst individuals with an affected relative [e.g. (Austin et al., 2006; Meiser et al., 2008)] and amongst the general population unselected for disease risk (Cameron et al., 2009; Laegsgaard et al., 2009; Wilde et al., 2011), few data describe anticipated health behaviours as a consequence of such testing. How individuals respond to genetic risk is especially complex when penetrance and predictive power of genotype are uncertain. The issue is further complicated by knowledge that a genetic component only represents part of the risk for

* Corresponding author at: School of Psychiatry, University of New South Wales, Black Dog Institute Building, Hospital Road, Randwick, NSW, Australia 2031. Tel.: +61 2 9382 9249; fax: +61 2 9382 8151.
E-mail address: alex.wilde@unsw.edu.au (A. Wilde).

0165-0327/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.jad.2011.06.023

multifactorial disease and appropriate behavioural responses to environmental risk factors are also required to make health behavioural interventions effective.

Psychiatric genetic epidemiological studies such as those reported by Kendler and Karkowski-Shuman, 1997 have consistently reported significant gene × environmental interactions in the genesis of depression. Caspi et al., 2003 have previously reported that the s/s genotypic variant of the serotonin transporter gene was associated with increased risk for major depressive disorder in interaction with stressful life events. Although the validity of this molecular association remains controversial, with both positive and negative meta-analyses reported (Caspi et al., 2010; Karg et al., 2011; Risch et al., 2009), the overarching premise of increased genetic and environmental risks leading to major depressive disorder formed the rationale for this study. Moreover, our previous study (Wilhelm et al., 2009), found that participants who carried the higher risk (s/s) variant and were provided with this information ranked 'earlier intervention and potential to prevent the onset of depression' as the highest perceived benefit of being provided with their genetic risk status.

The marketing of an increasing range of genetic tests for psychiatric disorders direct-to-consumer (DTC) (Hudson et al., 2007) without medical supervision, raises concerns about the psychosocial impact of risk disclosure and health behavioural outcome of such genetic risk information. Several companies are currently marketing DTC genetic tests for predisposition to major depressive disorder (Genetics and Public Policy Center and Johns Hopkins University, 2010) and for the purposes of predicting individual response to selective serotonin reuptake inhibitor antidepressant (Mayo Clinic, 2006). There is strong evidence that genetic risk information impacts on perception of disease, which in turn has implications for health behaviours that aim to modify environmental risk factors (Senior et al., 2000). It has also been argued that provision of information about individual genetic risk alone may not be sufficient to change health-related behaviour (Javitt, 2006; Lemke, 2004; Marteau and Lerman, 2001).

Using hypothetical genetic susceptibility to major depressive disorder as an example, the present study aims to assess preparedness to modify risk for major depressive disorder at a pre-symptomatic stage through a range of preventive behaviours. This is the first national population study to examine this issue for genetic risk associated with mental health in general. The present study tested the following hypotheses: Willingness to engage in health behaviours that could ameliorate risk for major depressive disorder based on a hypothetical genetic susceptibility will be positively associated with i) a personal history of a mental illness, ii) self-estimation of risk for major depressive disorder as higher than the average person, and iii) endorsement of gene–environment interaction as a causal mechanism for mental illness.

2. Methods

Participants across Australia were recruited by a contracted market research company in May 2008 using random digit dialling of a computer-generated list of landline phone numbers that uses prefixes based on the geographic coverage of the sample's area, with the aim of producing a nationally representative sample. Respondents were selected from each household using a Computer Assisted Telephone Interviewing (CATI)-generated algorithm. Only those aged 18 years or more, and fluent in English were eligible to participate. Only one individual per household could participate. A target sample size of at least 1000 completed CATI interviews was reached. Ethical approval for the study was provided by the relevant Institutional Review Board.

This survey and sample have been previously described in a prior publication by our group (Wilde et al., 2011) which reported community interest in predictive genetic testing for susceptibility to major depressive disorder. In the current paper, we examine the willingness of these participants to engage in health behaviours that could ameliorate risk for major depressive disorder based on such hypothetical genetic susceptibility.

2.1. Demographic characteristics

Data on sex, age, highest level of education achieved and current marital status were collected using specifically designed multiple-choice items.

2.2. Self-estimation of risk for major depressive disorder

Data on self-estimation of risk of depression were collected in a three-part question early in the survey: 'Compared with the average person, would you say your risk of depression is higher than average; lower than average; the same as the average person?'

2.3. Clinical and family history data

Self-reported data on personal history of mental illness or exposure to others' experiences of mental illness through close relatives or close friends were collected on completion of the survey. Participants were asked 'have you' or 'has a close relative or friend ever been diagnosed with depression, bipolar disorder or schizophrenia?' These terms were defined to participants.

2.4. Causal attributions for mental illness

To assess the perceived importance of different factors in causing a mental illness a list of potential contributing factors were derived from Meiser et al., (2007). These were 'genetics'; 'accumulation of daily life stresses'; 'imbalance of chemicals in the brain'; 'major life changes'; 'being in a difficult relationship or marriage'; 'personality factors': 'a difficult or abusive childhood;' 'sexual abuse'; 'recreational drug abuse'; 'family environment'; 'parental behaviour'; Participants were asked: 'how important is…[insert item]… as a cause of mental illness?'

Participants responded to all items using a five-point Likert-type scale ranging from 1 'Not at all important' to 5 'Extremely important'. For statistical analysis, items were grouped according to an exploratory factor analysis which yielded a four factor solution with good internal consistency with item groupings representing (i) genetics, (ii) life stress, (iii) abuse and (iv) family environment (Meiser et al., 2007).

Three items with five-point Likert-type response options ranging from 1 'Strongly disagree', to 5 'Strongly agree' were used to assess endorsement of perceptions about causal

Download English Version:

https://daneshyari.com/en/article/6235160

Download Persian Version:

https://daneshyari.com/article/6235160

Daneshyari.com